Studies of host genetic risk factors to viral diseases describe how our genes affect the risk of being infected, or to develop disease after infection with a specific virus. With increased knowledge of why susceptibility to infectious diseases differs between individuals it may be possible to improve treatment and to foresee who are at risk for a certain infection. However, a genetic setup that makes an individual resistant to one pathogen can make him or her more susceptible to another - like a double-edged sword.

In this thesis I describe two viruses. In the first part I illustrate why only some of us are infected during an outbreak with norovirus, the virus causing the yearly appearing “winter vomiting disease”. I also describe a norovirus strain, which has circumvented the factor that normally prevents the virus to infect parts of the population.

The other virus discussed in the thesis is tick-borne encephalitis virus (TBEV). TBEV is, as the name implies, transmitted to humans from ticks and while most infections are asymptomatic, some patients develop tick-borne encephalitis (TBE), with severe symptoms including meningitis or encephalitis. We have found two host genetic factors involved in the immune response, which affect the risk of TBE.
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